ARTICLE XIII.

ACTION OF THE CILIARY MUSCLE IN ASTIGMATISM.

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CAN regular astigmatism be partly or wholly corrected by an unequal contracture of the ciliary muscle? This question was answered in the affirmative some ten years ago by Dobrowsky, and his opinion has since been generally adopted. It is rare, however, that so positive a proof of it can be furnished as by the following case:—

A. S., 17 years of age, had complained of ocular fatigue and even slight pain for many years. One year ago he obtained a pair of concave cylinders 24, but the old trouble was but little alleviated. On examination I found a convergent strabismus of the left eye, with a slightly paretic condition of the external rectus. On covering the other eye the usual secondary deviation occurred, slightly more excursive, than the primary deviation of the left eye, while both eyes performed oscillatory movements. This nystagmus I could time very easily; it consisted of a comparatively slow movement of the left eye towards the right side (action of rectus internus), alternating with a rapid jerk to the left, in order to bring the visual axis again into the desired direction. The movements of the other (not squinting) eye were the same in direction and evidently secondary, *i. e.*, due to association. This nystagmus occurred only when the right eye was covered; its cause must be sought in the insufficiency of the paretic external rectus of the left eye. The directions of these ocular movements and of the *apparent* motion of external objects resulting from the nystagmus, corresponded with the ocular phenomena due to vertigo or lesions of the semicircular canals.[1] Omitting further discussion of these motor anomalies, as well as all irrevelant details, I can describe the optic condition of the left (strabismic) eye as a myopic astigmatism of about 10 dioptrics; accurate determination being impossible on account of the diminished visual acuity (V not quite $\frac{20}{200}$). The sight of the right was also less than normal, its corrected V being $\frac{20}{80}$, while the apparent refraction was M 2 D with As M 5 D. After a thorough use of atropia for two days, I re-examined and found the correcting glass for the left eye equal to cylinder —10 dioptrics. But since the amblyopic eye did not take part in binocular vision, all further remarks apply only to the right eye, the correcting glass of which, when atropinized, was found to be cylinder —6 D, axis horizontal. There was, besides, considerable irregular astigmatism, which could only be remedied by a stenopaic slit.

A fortnight later Mr. S. returned, complaining that he could not use his glasses for reading, although reading was possible with his former spectacles (cyl. —24), and even with the naked eye. For the distance his glasses gave perfect satisfaction, his sight had even improved (V $\frac{20}{60}$), since he had learned to ignore the circles of dispersion due to the irregular astigmatism. The effects of atropia had completely disappeared as far as the iris was concerned, and on removing his glasses I could satisfy myself that his power of accommodation was the same as before the use of atropia. It seemed to me most likely that his inability to read with his correcting glasses was owing to a *partial* contraction of the ciliary muscle, tending by itself to correct his astigmatism, and hence rendering the glass, which was exact for the distance, too strong for the near point. I tested this view with some large print, which the patient read distinctly at a distance with his glass. On bringing the paper nearer to the eye, it began to get blurred at a distance of one metre, the indistinctness increasing with the proximity to the eye. In order to maintain distinctness, it was found necessary to diminish the strength of his correcting glass (cylinder —6 D, axis horizontal) by neutralization with convex cylinders in the ratio of the proximity of the object. Thus at one metre distance the strength of his glass had to be reduced by one dioptric, while with an object 25 centimetres off a convex cylinder of 4 D was required to neutralize the excessive strength of his own glass. While the ciliary muscle was completely at rest on correcting the eye for the distance, there occurred—evidently from previous habit—a partial contraction (of those fibres only influencing the horizontal meridian of the lens) on attempting to see close by. In the absence of any glass, *the eye would have corrected in part its own astigmatism, the degree of correction increasing with the proximity of the object.* The inability to contract the ciliary muscle uniformly was shown by the following crucial test. An emmetropic eye can see plainly in the distance through any concave glass, which it can overcome by its own accommodation. But this patient, with his eye corrected for the distance, could *not* see distinctly through a concave glass of 4 dioptrics. Instead of an accommodative effort equal to a spherical glass of 4 D, his ciliary muscle increased the refraction of the *horizontal* meridian only, and in order to see distinctly, it was necessary to use an additional *convex cylinder of* 4 *D, axis horizontal* (or in other words to diminish his correcting glass from a concave cylinder of 6 D, axis horizontal, to 2 D, the remnant of his astigmatism being neutralized by his own accommodation).

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[1] *Vide* H. Gradle, Chicago Med. Journal and Examiner, Aug. 1878.

My assurance, that the patient would gradually learn to contract his ciliary muscle uniformly, and get his eye accustomed to the full artificial correction of its astigmatism, proved correct. One week later reading became easier, and, testing him with glasses, only weaker convex cylinders were required to neutralize the excessive strength of his own glass for the near point. Within about six weeks all trouble had disappeared, and the mode of ciliary contraction had become normal.

In the above description I have referred to ciliary contraction as the cause of accommodation for the near point, notwithstanding the objections urged by Dr. M. J. De Rosset. In the last number of this Journal Dr. De Rosset attempted to overthrow the usually accepted theory of accommodation, substituting for it the statement, that accommodation for the near point is the result of *relaxation* of the ciliary muscle, while the adjustment of the eye for distance depends on the contraction of the same. His main arguments, however, belong to the class of "circumstantial evidence," which must receive an interpretation, in order to be utilized; and the interpretation according to the Helmholtz theory appears natural. But apart from indirect arguments, the theory of Helmholtz is directly proven by the experiments of Hensen and Völkers. Every excitation of the ciliary ganglion or nerves caused in their experiments an accommodative movement. This they demonstrated by needles introduced as levers through the sclerotic. The needle piercing the ciliary body, and the one passing through the fovea centralis did not move, the others did, demonstrating, *that a ciliary contraction pulls the choroid forward.* Other needles acted upon as levers by the surfaces of the lens showed, that with increasing convexity the anterior surface of the lens advances considerably, while the posterior surface recedes very slightly. A last lever finally proved the advancement of the zonula with every accommodative effort. The movements of the lens could even be observed directly on ablation of the cornea. These observations pertain not only to the eyes of the dog, cat, and monkey, but were also confirmed on an extirpated human eye. They establish positively the following as the order of cause and effect: nerve-excitation, contraction of the ciliary muscle, advancement of the insertion of the zonula, and hence its relaxation, increasing convexity of the lens.

The doubts which De Rosset expresses as to the more recent experiments of Hensen and Völkers do not seem justified. Whenever the experiment did succeed, they obtained an accommodative movement from excitation of the trunk of the motor oculi. The want of success in many experiments does not surprise any one familiar with the mutilations incident to, and the difficulty of experiments on, intracranial nerves. The belief that any other nerve is concerned in the function of accommodation has no experimental or clinical basis. In previous experiments Trautvetter had also failed to obtain an accommodative response from excitation of the third nerve in mammals, probably on account of similar difficulties. In birds, however, accommodation for the near point was obtained by stimulation of the motor oculi.

The positive evidence of these experiments once accepted, the further arguments of Dr. De Rosset lose their validity.[1]

[1] The last number of the *Centralblatt f. d. Med. Wiss.* (No. 43, 1878) contains a communication by Dr. J. Hock, of Vienna, which fully corroborates the experiments of Hensen and Völkers, on stimulation of the short root of the ciliary ganglion (the motor oculi nerve). In curarized dogs he could easily observe the increase of convexity of the anterior surface of the lens as manifested in the changed size of the reflected image of a calcium light. In order to keep the pupil dilated during excitation of the third nerve, he resorted ingeniously to simultaneous excitation of the sympathetic. Needles piercing the sclerotic showed further that a ciliary contraction pulls the choroid forward, but does not act directly on the vitreous body. All manifestations of contraction of the ciliary muscle were completely prevented by an energetic application of atropia.